PERINATAL OUTCOME IN MECONIUM STAINED AMNIOTIC FLUID

Dissertation submitted for

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BONAFIDE CERTIFICATE

This is to certify that the study entitled "**Perinatal Outcome in Meconium Stained Amniotic Fluid**" is the bonafide work done by **Dr.G.Indhumathi,** at the Institute of Obstetrics and Gynaecology, Govt. Hospital for Women and Children attached to Madras Medical College, Chennai during the period of her Post Graduate study for MD Branch II Obstetrics and Gynaecology, from 2003 – 2006 under the guidance of **Prof.Dr.K.Saraswathi, M.D. DGO., .**

This dissertation submitted to Dr.M.G.R. Medical University is in partial fulfillment of the University rules and regulations for the award of MD Degree in Obstetrics and Gynaecology.

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INTRODUCTION

The birth process is described as the most perilious journey an individual ever undertakes. Meconium staining of the amniotic fluid and abnormalities in the fetal heart rate pattern have long been recognized as danger signals in this journey.

J. Whitridge Williams (1903) observed that “characteristic sign of impending asphyxia is the escape of meconium”. There are two principal reasons why meconium is passed by the fetus-maturity and fetal compromise. Meconium passage reflects maturity of the central nervous system and the gastrointestinal tract. Incidence of meconium stained liquor increases steadily from 10% at 36 weeks to 30% at 40 weeks and 50% at 42 weeks. Fetal compromise of an acute or subacute nature also leads to passage of meconium. Whatever be the controversies regarding meconium, the following holds true:

1. Clear amniotic fluid is reassuring.

2. Thick fresh meconium in a situation of high risk is of great concern.

Presence of abnormal fetal heart rate pattern in the presence of meconium stained amniotic fluid is a definite indication of fetal compromise.

AIM OF THE STUDY

This prospective study aims :

1. To evaluate the perinatal outcome in term pregnancies with meconium stained amniotic fluid.

2. To bring out the correlation between fetal heart rate abnormalities and perinatal outcome in meconium stained amniotic fluid.

REVIEW OF LITERATURE

"The birth of a healthy baby is the universal aim".

HISTORICAL PROSPECTIVE:

Meconium is a term derived from the Greek word 'Mekonion', a word for 'poppy juice' or 'opium like'. Aristotle is credited with having drawn the analogy between the presence of this substance in the amniotic fluid and the "sleepy" newborn.

Laennec (1806), a physician in Paris was the father of technique of auscultation of adult heart and lungs.

Schwartz and Von Winckel (1858) stressed the importance of fetal heart rate auscultation throughout labour. He thought the appearance of meconium in labour meant impending fetal death.

In ***1925, Schulze*** conducted a study of 5500 births in California and concluded that passage of meconium during labour is in the large majority of cases independent of fetal asphyxia and the presence of old meconium in the amniotic fluid was of no prognostic significance for the later development of asphyxia. She has also observed that in cases associated with asphyxia, there were always changes of the fetal heart rate pattern during labour.

In ***1958, Caldeyro Barcia, Hon and Hammacher*** reported their observation on various fetal

heart rate patterns associated with fetal distress.

Ferton and Steer (1962) suggested that passage of meconium was significant only if fetal heart rate was less than 110 bpm.

In ***1966, Saling*** introduced amnioscopy for pregnancies more than 10 days past the expected date of confinement and suggested that the finding of meconium indicates impending danger and immediate amniotomy and fetal blood sampling should be performed. He postulated that fetal hypoxia precipitates fetal gut vasoconstriction which causes hyperperistalisis and sphincter relaxation with passage of meconium.

Brandes et al, (1973) observed that fetuses who have passed meconium during labour are in a state of temporary compensated fetal distress and should be delivered within a reasonable time.

Miller et. al., (1975) found no difference in neonatal Apgar between meconium and non-meconium group if the fetal heart rate during labour had been normal. They concluded that the presence of meconium in the absence of other signs was not a sign of fetal distress.

Meis PJ; Hall. M(1978) observed that thin meconium stained amniotic fluid was not found to be associated with any increased intrapartum or neonatal morbidity or mortality in contrast to thick meconium stained amniotic fluid.

Starks et al., (1980) found that thick meconium was associated with lower fetal scalp blood pH than thin or absent meconium and concluded that thick meconium usually indicates fetal hypoxia or

acidosis regardless of abnormal fetal heart rate. However more recent studies have established that infants with normal heart rate patterns have similar outcome whether or not meconium is present in the amniotic fluid.

Krebs and Coworkers (1980) concluded that bradycardia and deceleration are significantly increased in patients with meconium stained liquor. He devised an intrapartum cardiotocographic scoring system.

Benacerraf et. al., (1984) reported that the detection of thick meconium by ultrasonography, but further studies showed that vernix can produce a similar picture.

Grant et al., (1989) concluded that using a low 5 minute APGAR score as endpoint (APGAR < 7) abnormal fetal heart rate has a high negative predictive value of over 90% but a low positive predictive value of 30%. This means that normal trace indicates a fetus is not hypoxic but abnormal trace is associated with large number of false positives.

Steer PJ, Eigbe F, Lissauer TJ, Beard RW (1989) conducted a large study on 1219 patients with meconium stained amniotic fluid monitored by cardiotocography and sensitivity was 80% at any time for acidosis and predictive value was 32%.

Lately, studies on Urinary Meconium Index (UMI) by spectrophotometry have been reported. The entry of meconium into the maternal circulation occurs during labour pains and may be excreted in the mother's urine. The entry takes place even in the absence of any clinical signs of rupture of membranes. Patient who delivered babies with low Apgar had higher positive UMI of rising type.

(Chinese Journal of obstetrics of Gynaecology 1990).

In ***1993 Steer PJ*** and ***Smith R*** studied the continuous monitoring of meconium in liquor by optical sensor mounted to an intrauterine probe.

SIGNIFICANCE OF AMNIOTIC FLUID MECONIUM

Meconium is a viscous green liquid that consists of GIT secretions, bile, bile acids, mucus, pancreatic juice, cellular debris, amniotic fluid, swallowed vernix caseosa, lanugo hair and squamous cells. It is rarely seen in the amniotic fluid until mid to late 3rd trimester. The incidence of meconium staining of the amniotic fluid is approximately 10% of all pregnancies. In 35% of these meconium is aspirated into the fetal lung and 10-40% of the asphyxiated babies who aspirate die neonatally.

FORMATION OF MECONIUM:

The gastrointestinal tract originates from both endoderm and splanchnic mesoderm by day 14 after fertilization and is lined by undifferentiated cuboidal cells by day 18 ***(Arey, 1974; Grand et al., 1976)***. Intestinal villi appear by 7 weeks and active absorption of glucose and aminoacids occurs at 10 weeks and 12 weeks respectively. By 12 weeks gestation, development of Meissner's and Auerbach's plexuses within the intestinal wall coincides with onset of peristalisis of the small intestine and colon. Meconium appears in the fetal intestine at approximately 70-85 days gestation ***(Smith, 1976).*** High concentrations of intestinal enzymes are present in amniotic fluid early in gestation followed by a decline that could be related to increased anal sphincter tone ***(Potier et al., 1978 and Mulivor et. al., 1979).***

Composition of meconium:

Colour	:	Dark green.
Physical properties	:	Thick, viscous and odourless.
Dry weight	:	28%
Protein	:	No demonstrable amount.
Carbohydrate	:	80%.
Lipid	:	Minimal.
Blood group substances	:	Present.
Nitrogen	:	High
pH	:	5.5 to 7
Electrolytes	:	Na, K, Ca, Mg, Cu, Zn,
Water	:	72 – 80%

Meconium also contains bile acids and salts, enzymes, amniotic fluid, swallowed vernix caseosa, lanugo hair and squamous cells. Large concentrations of bile pigments excreted by the biliary tract from the fourth month onward give meconium its green colour. The fetus lacks intestinal bacteria, which accounts for many of the differences in composition between meconium and adult stool.

Theories of meconium passage :

Maturation theory:

Because meconium seldom is observed preterm ***(Scott et al., 2001)***, its presence in amniotic fluid could reflect gastrointestinal maturity in late gestation. ***(Matthews and Warshaw, 1979)***. The hormonal control of fetal meconium passage is maturation dependent. Motilin, an intestinal peptide responsible for bowel persistalisis and defecation is high in the umbilical cord of term infants who have passed meconium compared to preterm with clear liquor.

The neural control of meconium passage is also dependent on gestational age because maturation and myelination of gastrointestinal tract progresses throughout gestation. Immaturity of intrinsic and extrinsic innervation of the bowel would impair the ability of premature fetus to pass meconium into the amniotic fluid. At autopsy, preterm neonates have more unmyelinated nerve trunks and fewer ganglion cells in distal colon compared with term neonates.

Transit time through fetal small intestine decreases as gestation advances. Further more as the fetus matures, the intestinal tract, becomes more responsive to sympathomimetic agents. Parasympathetic stimuli initiate meconium passage after maturation of fetal intestinal tract after 34 weeks. The incidence of meconium passage during labour increases with the gestational age and reaches approximately 30% at 40 weeks and 50% at 42 weeks.

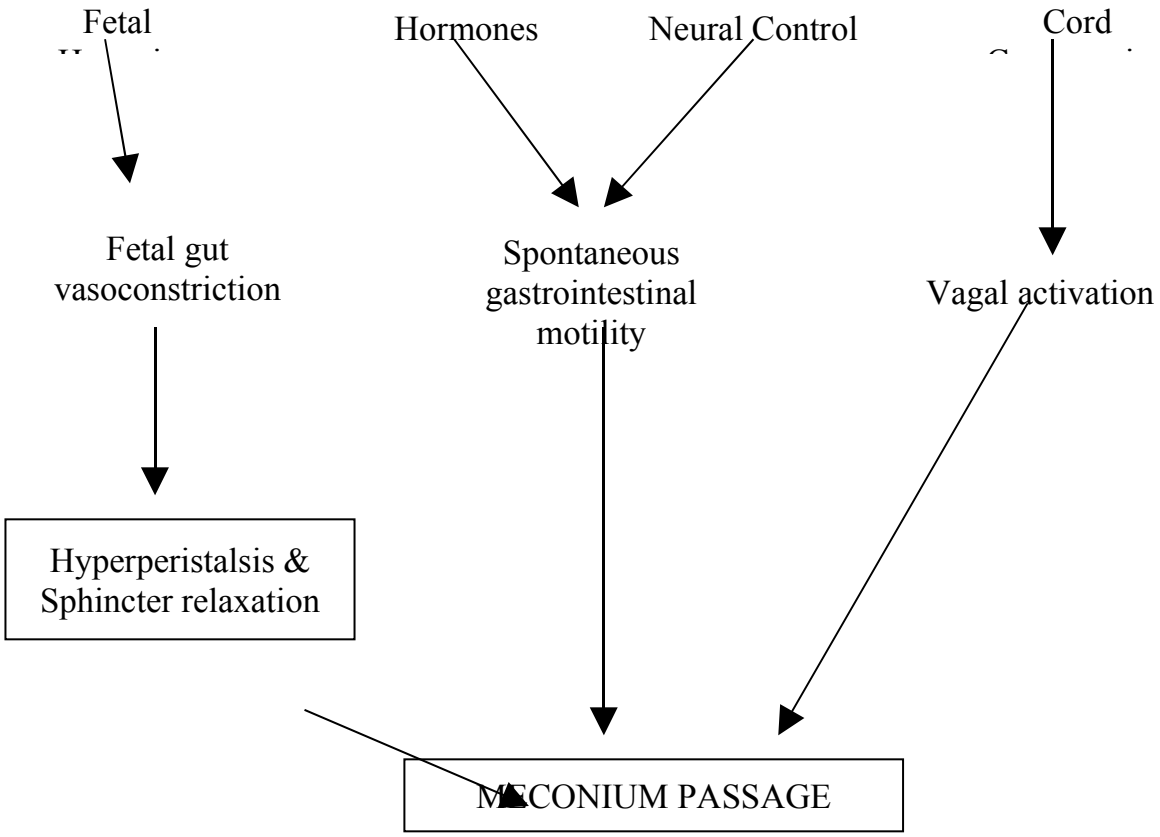
Theory of fetal distress:

The relationship of fetal hypoxia and intestinal persistalisis has been a consideration for many years. ***Walker (1954),*** demonstrated that meconium was released more frequently when the oxygen saturation of the umbilical vein was below 30% and that heavy meconium is associated with lower oxygen saturation more often than light meconium. ***Hon (1963)*** suggested that meconium is passed in response to parasympathetic stimulation during cord compression, but ***Krebs*** and ***Associates (1980)*** found no difference in the frequency of variable decelerations regardless of whether meconium was present. Umbilical cord erythropoietin concentrations are elevated in human pregnancies complicated by meconium stained amniotic fluid, suggesting an association between chronic hypoxemia and meconium passage ***(Richey et al., 1995; Jazayeri et al., 2000). Manning and Coworkers (1990)*** reported that amniotic fluid meconium was present more than twice as often if the last biophysical profile score was abnormal (6 or less).

Fetal compromise usually of an acute or subacute nature leads to passage of meconium. There are various degrees of meconium staining from diluted old meconium which is brownish yellow to thick green “pea soup” meconium. Typically thick undiluted meconium seen in breech presentation is for obvious mechanical reason. Meconium passage in preterm infants can occur if it becomes infected with organisms which can cause a fetal enteritis (Listeria monocytogenes, ureaplasma urealyticum, rotavirus).

Thick meconium stained amniotic fluid is associated with increased peripartum infection rates. Some reports have suggested an increased risk of meconium passage in association with cholestasis of pregnancy.

ETIOLOGY OF MECONIUM PASSAGE



Meconium and fetal distress

Is meconium, a marker for fetal distress ?

Obstetric textbooks though the 17th, 18th and 19th centuries reported meconium passage as a sign of fetal death or impending death.

However it is not true to say " no death without defecation". As Miller et al., have stated, the presence of meconium in the amniotic fluid without signs of asphyxia is not a sign of fetal distress, and is not in itself, a cause for intervention. ***Katz and Bowes (1992)*** stated that "when normal FHR patterns are found with meconium stained amniotic fluid the neonatal outcome is similar to neonates with clear fluid. Similarly, in infants with meconium stained amniotic fluid with antepartum signs of distress – neonatal outcomes are similar to those of non – meconium stained infants with similar FHR abnormalities". Perhaps the most important clinical value of meconium stained amniotic fluids is to alert the obstetrician, to look for further signs of fetal compromise.

Meconium aspiration syndrome (MAS)

The term "meconium aspiration" refers to the presence of meconium below the vocal cords and in the lungs. When amniotic fluid contains meconium, meconium is found below the vocal cords in approximately 1/3 of neonates, ranging from 21% to 56%. But meconium aspiration syndrome (MAS) develops in 2-8% of infants delivered through meconium stained amniotic fluids ***(Davies et al., 1985, Rossi et al., 1989).***

Aspiration of meconium was thought by many authors to occur at the time of delivery as the

new born infant take its first breath. So, oropharyngeal suctioning and tracheal toileting have been widely promoted in order to prevent MAS. However, it now seems likely that meconium aspiration most commonly occurs in utero as it still occurs despite adequate suctioning at delivery.

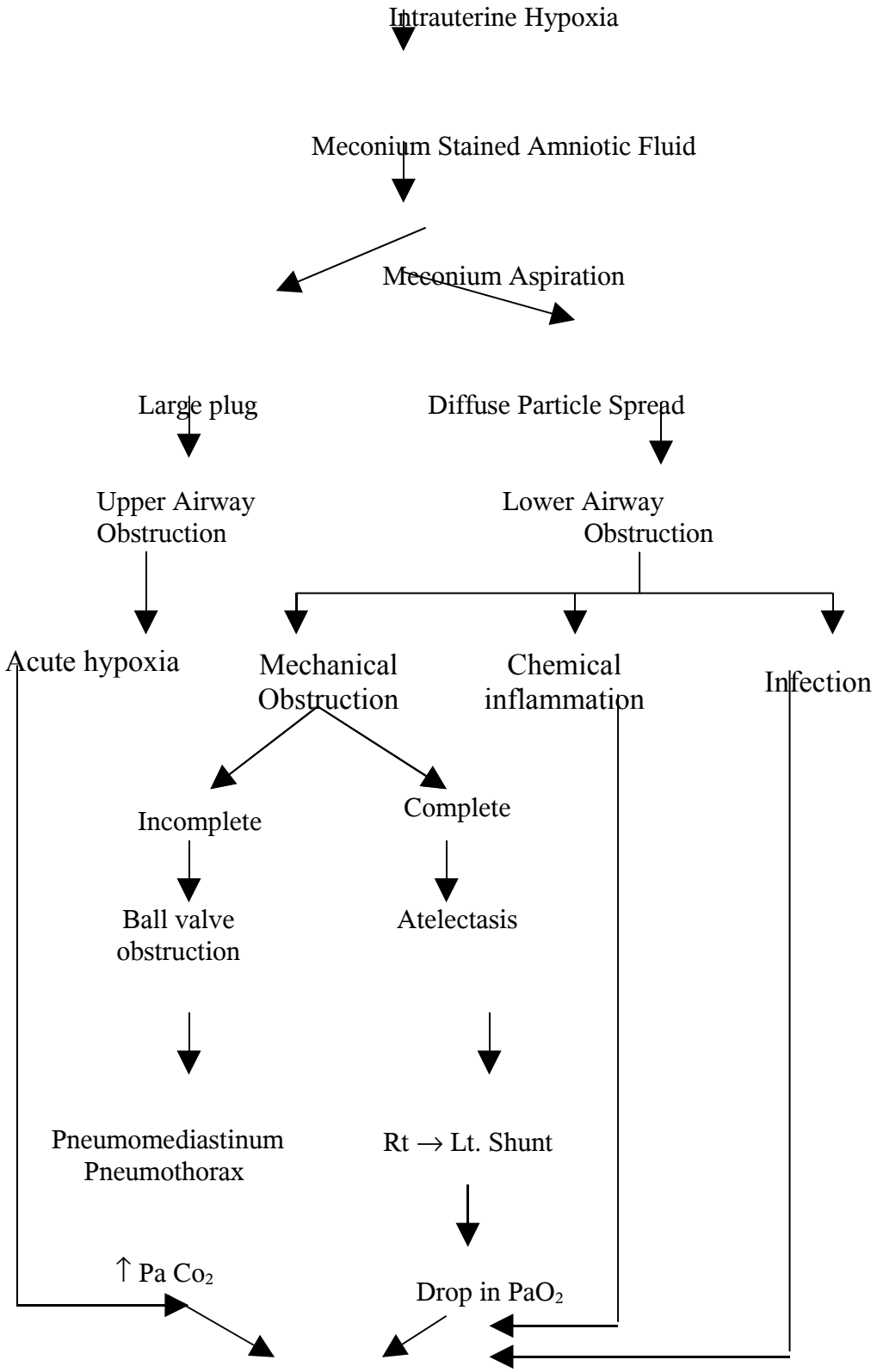
Meconium aspiration occurs in utero due to fetal breathing movements, which are of two types – gasping and deep breathing. Gasping is the fetal response to hypoxia. The fetus may also inhale meconium by deep irregular breathing in utero not associated with hypoxia. These breaths become more frequent as gestation advances and comprises about 10% of all fetal breathing movements. Fetal hypercapnia and acidosis also increase these breathing movements.

Meconium aspiration is more common when the meconium is thick rather than thin, this may be due to reflection of the fact that oligohydramnios and therefore undiluted meconium is more likely to lead to fetal hypoxia due to cord compression and consequently increased breathing.

PATHOPHYSIOLOGY OF MAS:

It involves a combination of mechanical obstruction and chemical pneumonitis of small airway by particulate meconium inhaled at the time of infant's first breath. ***(Tyler et al., 1978; Perlman et al., 1989)***

PATHOPHYSIOLOGY OF MECONIUM ASPIRATION SYNDROME



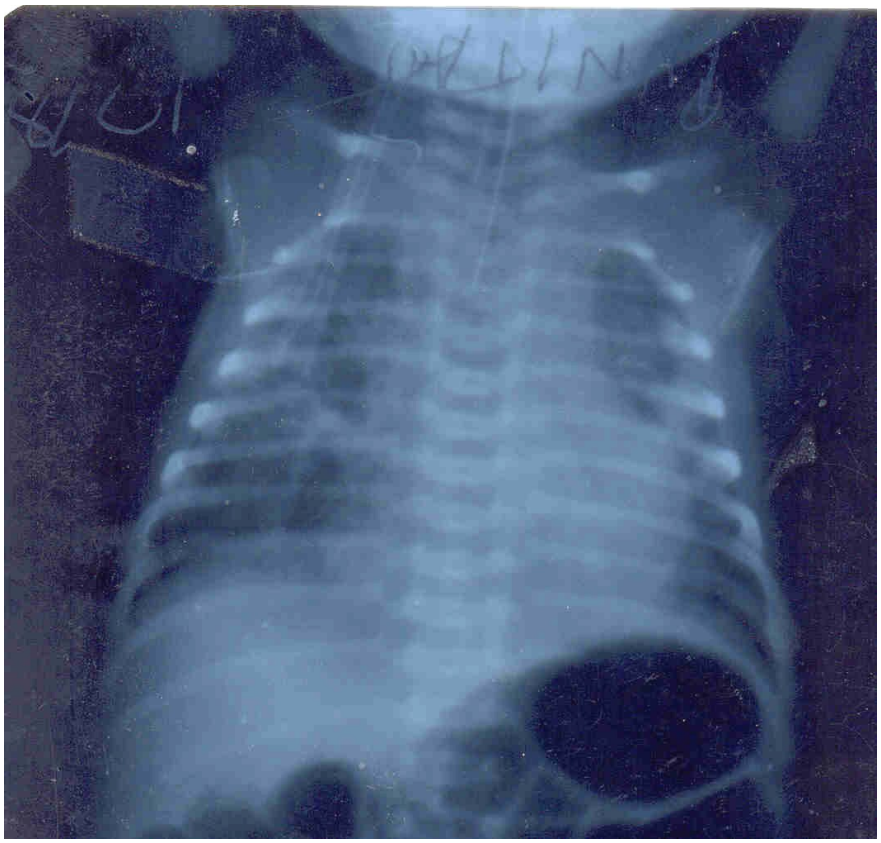
Hypoxia
Hypercapnea and
Acidosis

Resultant pulmonary vascular spasm then would lead to pulmonary hypertension and right to left shunting through the patent foramen ovale or ductus arteriosus. Worsening hypoxia could lead to convulsions, renal failure, disseminated intravascular coagulation and heart failure (Brady and Goldman, 1986). Pulmonary function can further be compromised by displacement of pulmonary surfactant by free fatty acids in meconium (Clark et al., 1987) or by direct inhibition of surfactant's surface tension-reducing properties, by meconium (oh and Bae, 2000; Hertig et al., 2001).

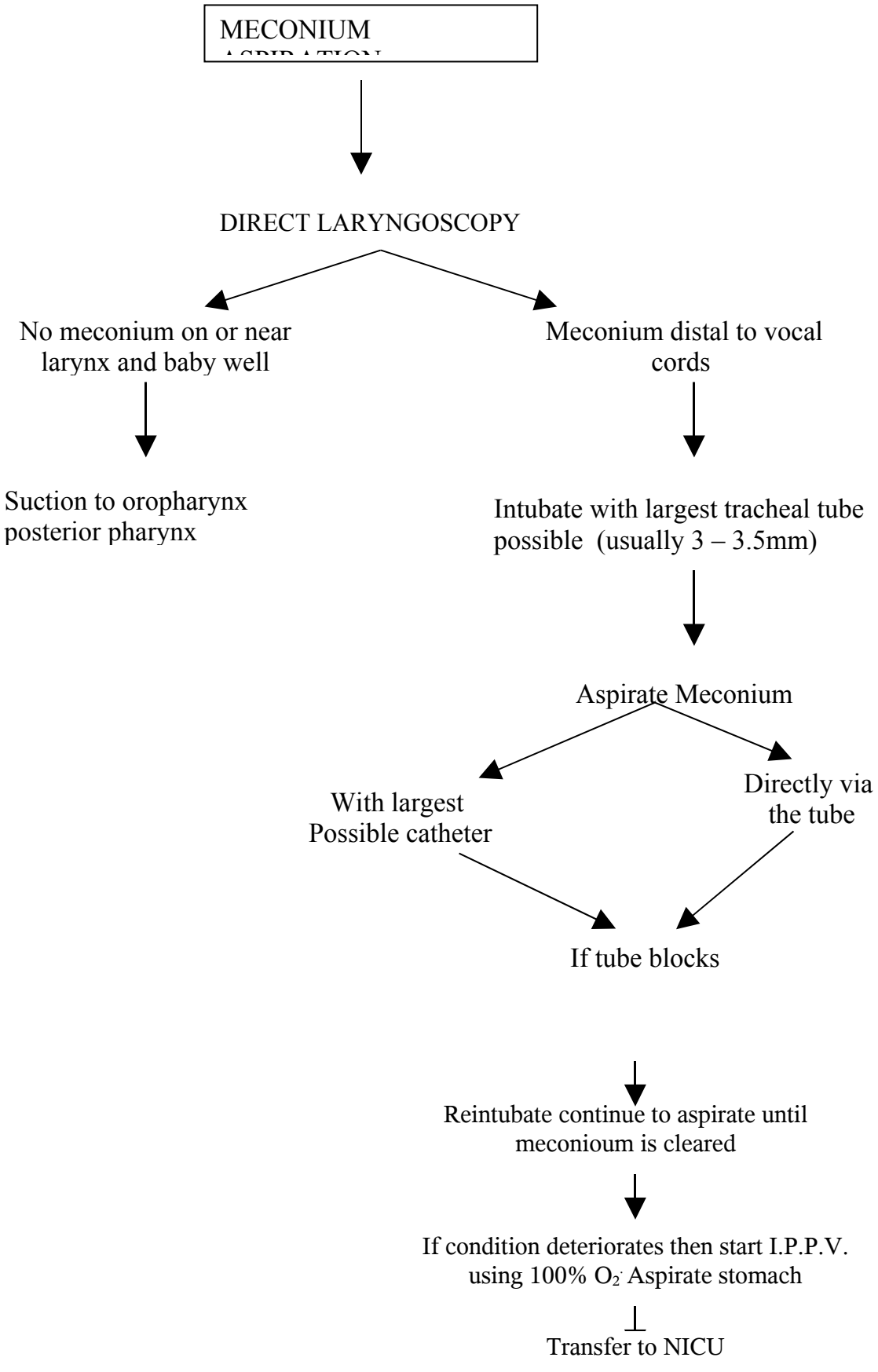
CLINICAL FEATURES:

MAS is characterized by mild to severe respiratory distress at birth. Meconium inhaled in an infant (which has not been subjected to hypoxia) usually causes mild disease only and is asymptomatic in 90% of cases. In its mildest form, the disease could present with neonatal tachypnoea associated with normal pH and low pCO2, resolving within 2-3 days. In its more severe form, the syndrome can present with hypoxemia, acidosis and respiratory failure.

CHEST X – RAY SHOWING MECONIUM ASPIRATION SYNDROME



MANAGEMENT OF MECONIUM ASPIRATION



Postnatal therapy for MAS begins with continuous observation and monitoring of infants at risk. Mechanical ventilation is required in upto 30% of infants with severe MAS and must be managed carefully. Continuous positive airway pressure or PEEP may aggravate hyperinflation associated with MAS and should be used with caution. The sickest neonates could even require extracorporeal membrane oxygenation (ECMO) to maintain oxygenation and prevent barotrauma until alveolar healing occurs and pulmonary function improves enough to meet baseline oxygen demands.

Pneumothorax or pneumomediastinum occurs frequently during the course of MAS because of the ball-valve effect of meconium and may occur before the application of positive-pressure ventilation. Therapy of the infant with MAS includes careful observation and vigorous treatment of other sequelae of neonatal asphyxia including temperature instability, hypoglycemia, hypocalcemia, hypotension, decreased cardiac function, reduced renal function, reduced liver production of clotting factors, hypoalbuminemia, cerebral edema and seizures. The mortality rate from MAS is as high as 40% in affected neonates.

PREVENTION OF MAS:

Is MAS preventable?

Pfenninger and Coworkers (1984) demonstrated that oropharnygeal suction is superior to nasal suctioning and although both methods should be used in succession.

Wiswell and Colleagues (2000) found that oropharyngeal suctioning before delivery of the thorax decreased the incidence of MAS, but there was no difference between bulb suctioning and De Lee suctioning.

Unfortunately, even the most vigorous suctioning before the first breath does not remove meconium already aspirated into the lungs before breath and thus does not eliminate the occurrence of MAS. Several authors have documented in utero meconium aspiration ***(Manning et al., 1978; Brown and Gleicher, 1981).*** The evidence supporting this contention comes from autopsy studies showing meconium in the alveolar spaces both in stillborn infants and in newborns vigorously suctioned before the first breath. Thus, MAS is not always preventable, despite aggressive airway management at the time of delivery.

Katz and Bowes (1992) questioned whether meconium is necessary for a reported diagnosis of MAS. Mild MAS follow from the classic theory of meconium inhalation, but severe MAS appear to be multifactorial.

Thickness of meconium and abnormal FHR patterns have been associated with a higher risk of MAS.

Rossi and Colleagues (1989) found that 19% of infants with thick meconium had MAS compared with 5% of those with moderate and 3% of those with thin meconium.

Fleischer and Colleagues (1992) reported that the risk of neonatal respiratory complications in the presence of meconium was 2% when FHR patterns were normal and 12% when they were abnormal. ***Paz and Associates (2001)*** found similar associations between MAS and FHR abnormalities.

Cunningham and colleagues (1990) and ***Yoder (1994)*** used routine oropharnygeal De Lee suctioning of all infants with meconium in the amniotic fluid, but tracheal intubation only for depressed infants with moderate to thick meconium.

In a large randomized trial (n = 2094) ***Wiswell and colleagues (2000)*** demonstrated no benefit to intubation and tracheal suctioning in vigorous infants born through amniotic fluid stained by any thickness of meconium.

The American Academy of Pediatrics (2000) recommended early oropharyngeal suctioning (before delivery of the shoulders) of all infants in the presence of meconium and tracheal visualization and suctioning for neonates who are depressed or born through particulate meconium.

MECONIUM STAINING OF THE TISSUES:

The degree to which tissues stain when exposed to meconium depends on the following:

1. Meconium concentration.

2. Length of exposure.

3. The nature of the exposed tissue.

Vernix suspended in meconium stains yellow in 12-14 hrs. Newborn fingernails stain yellow in 4-6 hrs. ***(Desmond et al, 1956)*** Miller and associates (1985) exposed placentas in vitro to meconium in varying concentrations and durations of exposure. Within one hour, surface staining was observed. Pigment accumulation in macrophages within the placenta, occurs by 3 hours. Umbilical cord staining occurred at 1 hour with 5% meconium and at 15 minutes with 10% meconium.

Meconium could have direct vosoconstrictive effects on the umbilical vein that result in vasospasm and impaired fetal placental blood flow ***(Altshuler and Hyde, 1989).*** In addition, meconium present for more than 16 hours occasionally induces umbilical cord ulceration and vascular necrosis, potentially compromising fetal oxygenation ***(Altshuler et al, 1992)***. These effects are due to bile acids present in meconium. Meconium in pulmonary macrophages suggests antepartum in utero aspiration. This information can be important postpartum in determining how long meconium has been present, especially in cases of meconium aspiration.

MECONIUM STAINED PLACENTA

AMNIOINFUSION

Amnioinfusion has been proposed as a way of diluting meconium and possibly decreasing the incidence of intrapartum MAS. It not only helps in diluting the meconium, but also in alleviating umbilical cord compression and variable fetal heart decelerations by replacing amniotic fluid volume, thereby restoring normal fetal oxygenation. These result would improve Apgar scores, raise umbilical artery pH and minimize inutero fetal gasping.

Here intrapartum transcervical amnioinfusion was considered after the detection of meconium either by spontaneous rupture of membranes or by amniotomy. The amnioinfusion catheter was inserted transvaginally and passed above the baby's head with one end outside. An intravenous drip was connected to this end and normal saline at room temperature was infused. Ideally, 1 litre of normal saline should be infused over 30-45 minutes.

In a randomized trial, ***Wenstrom and Parsons (1989),*** infused 1000 ml of normal saline through an intrauterine catheter into labouring women with fluid meconium and compared the outcomes of those women with those who received routine care. The amnioinfusion group experienced a six fold decrease in the incidence of meconium visualised below the vocal cords. Similar results have been reported by ***Sadovsky and Coworkers (1989); Macri et al, 1992 Cialone et al., 1994; Eiksen et al., 1994.*** A meta-analysis by ***Glantz and Hetteney (1996)*** concluded that amnioinfusion decreased the risk of meconium below the vocal cords by 84% ***Pierce and Colleagues (2000)*** and ***Hofmeyr (2001)*** also confirmed this reduction in MAS associated with amnioinfusion (relative risk approximately 0.25). Miyazaki and Navrez observed a relief of variable decelerations in 57% of patients receiving aminioinfusion as compared to 42% in non infusion group. So, amnioinfusion should be considered

when thick meconium is noted, especially when FHR monitoring is abnormal.

APGAR SCORE

Virginia Apgar (1953) developed the "APGAR SCORE" as a tool to assess the need for neonatal resuscitation. It was however designed to give an overview of fetal condition at set times following delivery, and to highlight those babies in need of resuscitation, not to define those babies with hypoxia. It is affected by other variables such as maternal opiate use, prematurity, aspiration of mucoid meconium, cardiac, respiratory, muscle, and CNS problems, and even in the hypoxic newborn does not give an indication of the time or duration of insult.

Sign	**Score**		
	0	**1**	**2**
Heart rate	Absent	< 100	> 100
Respiratory effort	Absent	Slow / irregular	Good
Muscle tone	Flaccid	Some flexion of extremities	Active motion
Reflex irritability	No response	Cry	Vigorous cry
Colour	Blue / pale	Body – pink with acrocyanosis	Completely pink

However in the past decade, low Apgar score has been considered as evidence that birth asphyxia has occurred and was predictive of abnormal neurological development in the offspring. Now the definitions of birth asphyxia used are based on Apgar scores, umbilical cord acid/base status, time to spontaneous breathing, and the neurological/behavioural condition of the infant. Using these definitions, the incidence of birth asphyxia lies between 2.9 and 9/1000 deliveries ***(Levene 1988)***.

The International classification of disease defines mild asphyxia as, a 1min Apgar score of ≤ 6 and severe asphyxia as a 1 min score of ≤3. However, in term neonates, 1 minute Apgar score was more influenced by the mode of delivery and by gestational age rather than by asphyxia. Instead 5 minute Apgar score had high concordance with metabolic acidemia. Very low late Apgar scores (0-3 at 20 min) were significantly related to mortality during the first year of life(96% in those < 2.5 kg, 59% > 2.5 kg). Low Apgar scores were only weakly related to morbidity as 80% of infants with a score of <3 at 10, 15 and 20 min that survived were without major handicap. Conversely of those babies developing cerebral palsy 55% had scores >7 at 1 min and 73% had >7 at 5 min.

Using a 5 min score of ≤ 7, Ruth and Raivio found the Apgar score to have a sensitivity of 12% and a positive predictive value of 19% for abnormal neurodevelopment at 12 months of age ***(Ruth & Raivio/1988).***

TERM INTRAUTERINE GROWTH RETARDED BABY

MATERIALS AND METHODS

This prospective study was conducted at Govt. Hospital for Women and Children, Egmore, (Institute of Obstetrics and Gynaecology) attached to Madras Medical College for a period from February 2004 to January 2006.

The study group consist of 300 pregnant women selected on the basis of inclusion and exclusion criteria. All of them had meconium stained amniotic fluid of varying degrees. The fetal heart rate abnormalities were recorded with intrapartum cardiotocography (Non – stress test). The mode of delivery and neonatal outcome were analysed.

The control group consist of 100 patients in labour with clear liquor.

STUDY DESIGN

Prospective Randomised Control Study

INCLUSION CRITERIA

1. Term gestation
2. Singleton pregnancy
3. Cephalic presentation
4. Primi or multigravida
5. With meconium stained amniotic fluid

6. Latent and active stages of labour
7. With or without risk factors (medical illness complicating pregnancy).
8. PROM

EXCLUSION CRITERIA

1. Multiple gestation
2. Malpresentations
3. Congenital anamolies of the fetus
4. Polyhydramnios
5. Antepartum hemorrhage
6. Preterm pregnancy

STUDY PROTOCOL

Patients in labour with meconium stained amniotic fluid were selected following the inclusion and exclusion criteria. Detailed history was taken and patients were carefully examined for any antepartum or intrapartum risk factors like preeclampsia, IUGR, APH, PROM etc. These patients were clinically monitored during labour. The colour of amniotic fluid and degree of meconium was noted at

the time of amniotomy or spontaneous rupture of membranes and at the time of delivery. The time interval between the detection of meconium and the time of delivery were noted. Fetal heart rate abnormalities were recorded using cardiotocograph, after the detection of meconium.

Depending upon the degree of meconium, fetal heart rate abnormalities, stage of labour and other risk factors, the time and mode of delivery was decided. After delivery, the immediate fetal well being was assessed by Apgar score at 1 min and 5 minutes.

Details such as cord around the neck, meconium staining of the cord, finger nails, vernix caseosa, meconium smearing of the body, caput, subgaleal bleed were noted. Weight of the baby taken into account. Evidence of IUGR, posmaturity and congenital anamolies were looked for.

After the delivery of placenta, look for its weight, meconium staining, infarcts, calcification etc.

Babies were followed up in the neonatal period upto 1 month using reply cards, for any morbidity and mortality. The morbidity criteria was taken as MAS, chest infections, fever and seizures.

OBSERVATIONS AND RESULTS

TABLE : 1

AGE OF PATIENTS

Age (Years)	Meconium group		Non – meconium group	
	Number of patients	Percentage	Number of patients	Percentage
16 – 20	12	4%	6	6%
21 – 25	120	40%	36	36%
26 – 30	120	40%	43	43%
31 – 35	48	16%	15	15%

$X^2 = 1.2$; $P = 0.76$

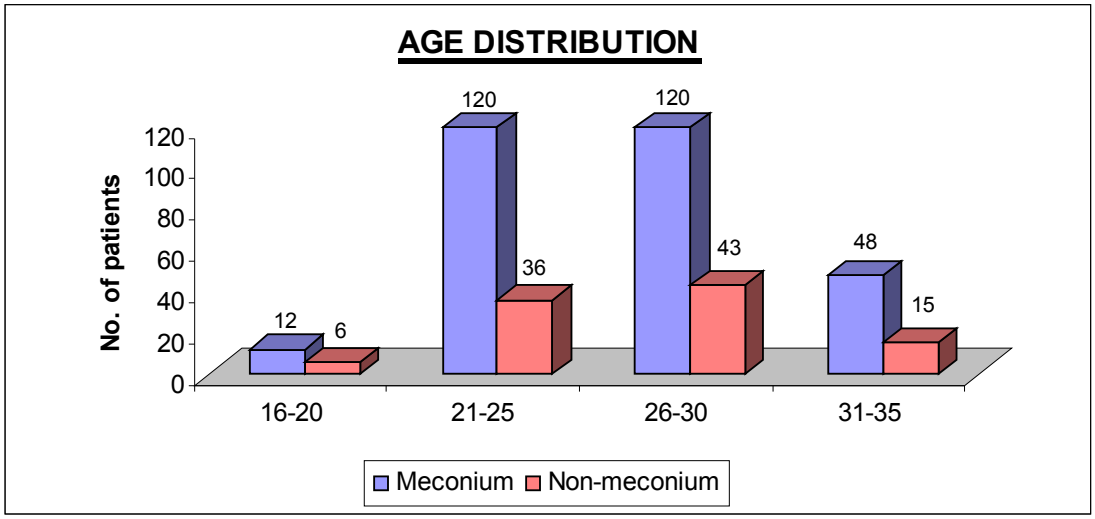
Majority of the patients were between 21 – 30 years of age in both the groups. In meconium group – 80% and in control group 79%.

TABLE 2

GESTATIONAL AGE

Gestational age (wks)	Meconium group		Non – meconium group	
	Number of patients	Percentage	Number of patients	Percentage
37 – 40	188	62.6%	65	65%
40 – 42	102	34%	34	34%
> 42 W	10	3.3%	1	1%

More than half of the patients were in the gestational age 37 – 40 wks both in study (62.6%) and control group (65%).



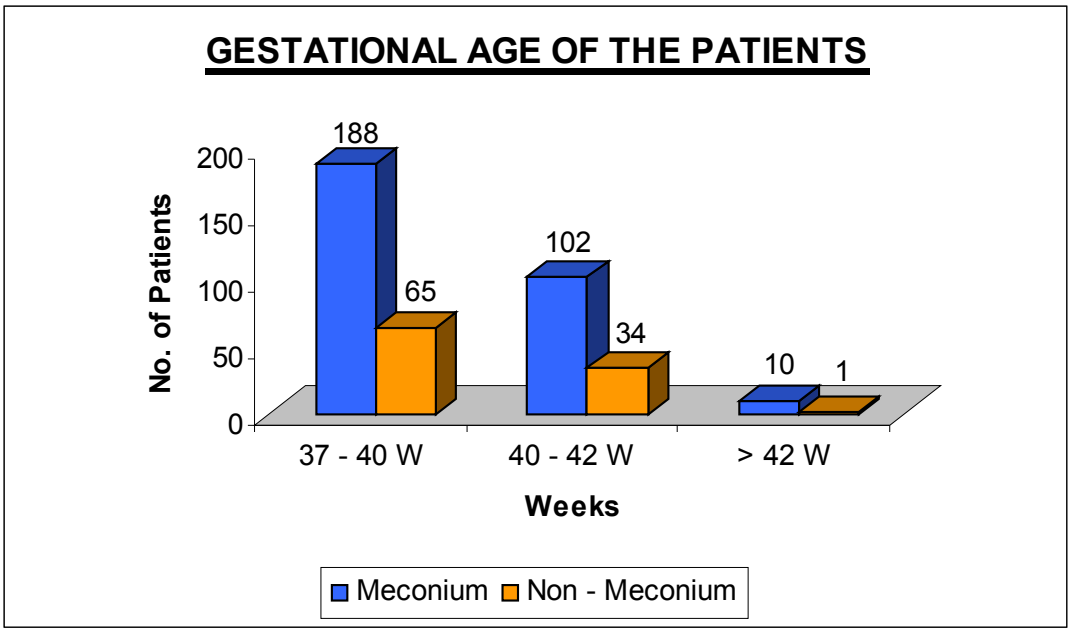

GESTATIONAL AGE OF THE PATIENTS
No. of Patients
200
150
100
50
0
188
65
102
34
10
1
37 - 40 W
40 - 42 W
> 42 W
Weeks
Meconium
Non - Meconium

TABLE – 3

PARITY OF THE PATIENTS

Gravida	**Meconium group**		**Non – meconium group**	
	Number of patients	**Percentage**	**Number of patients**	**Percentage**
Primi	183	61%	48	48%
Multi	117	39%	52	52%
Previous uterine scar	31	10.3%	14	14%

$X^2 = 5.4$; $P = 0.07$

In the study group, 61% were primigravida and 39% of the patients were multigravida.

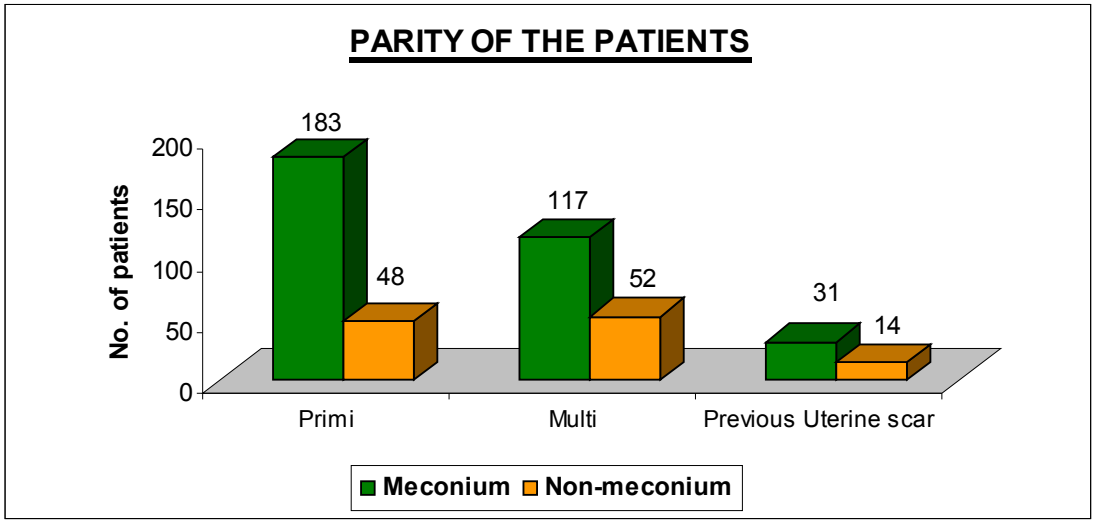

PARITY OF THE PATIENTS
No. of patients
200
150
100
50
0
183
48
117
52
31
14
Primi
Multi
Previous Uterine scar
Meconium
Non-meconium

TABLE – 4

BOOKING OF PATIENTS

Booking of Patients	**Meconium group**		**Non – meconium group**	
	Number of patients	**Percentage**	**Number of patients**	**Percentage**
Booked at IOG	159	53%	59	59%
Booked outside	126	42%	38	38%
Unbooked	15	5%	3	3%
Total	300	100%	100	100%

$X^2 = 1.45$; $P = 0.48$

Only 5% of the patients were unbooked in the study group.

TABLE - 5

CLASSIFICATION OF THE PATIENTS WITH RESPECT TO DEGREE OF MECONIUM

Density of meconium	**Number of patients**	**Percentage**
Thin meconium	105	35%
Moderate meconium	43	14.3%
Thick meconium	152	50.7%
Total	300	100%

Nearly half of the patients (50.7%) had thick meconium stained liquor.

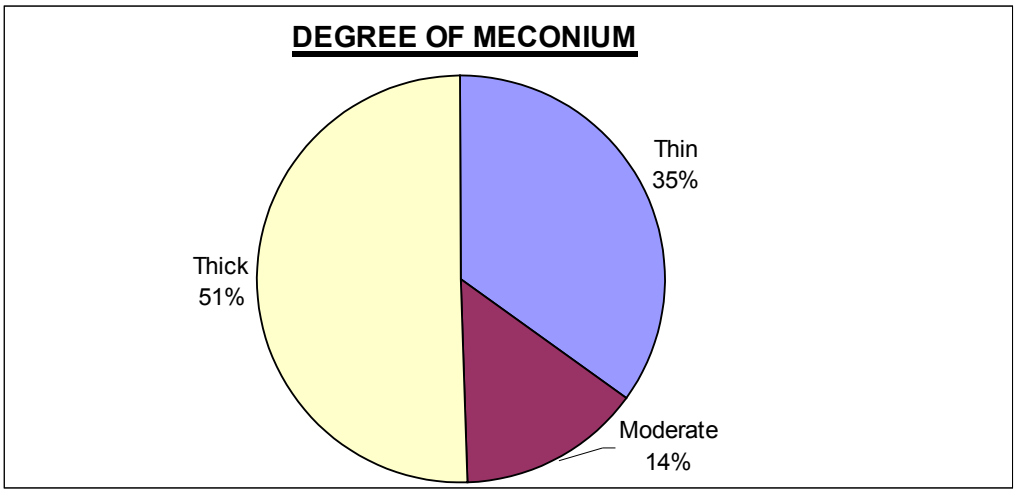

DEGREE OF MECONIUM
Thin
35%
Thick
51%
Moderate
14%

TABLE – 6

Stage of labour when meconium was detected

Stage of labour	**Number of patients**	**Percentage**
Latent phase	177	59%
Active phase	123	41%
Total	300	100%

In 59% of the patients meconium was detected in the latent phase of labour.

STAGE OF LABOUR WHEN MECONIUM WAS DETECTED

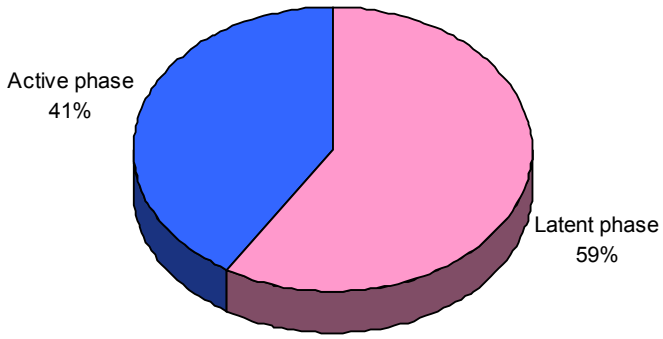


TABLE -7

TIME INTERVAL BETWEEN DETECTION OF MECONIUM AND DELIVERY

Density of meconium	**< 1 hour**	**1 – 3 hours**	**≥ 3 hours**
Thin MSAF	47	35	23
Moderate MSAF	20	19	4
Thick MSAF	126	24	2

$X^2 = 57.5$; P = 0.001

Majority of the cases of meconium stained liquor were delivered within 1 hour of detection of meconium.

AMNIOINFUSION:

Amnioinfusion was given for patients with moderate and thick meconium stained liquor. Among (43 + 154) 195 cases, amnioinfusion was given for 154 cases.

TABLE - 8

NST CHANGES IN MECONIUM GROUP WITH RESPECT TO DEGREE OF MECONIUM

Density of meconium	Reactive NST		Non reactive NST		Total number of patients
	Number of patients	%	Number of patients	%	
Thin meconium	78	74.28%	27	25.71%	105
Moderate meconium	19	44.19%	24	55.81%	43
Thick meconium	60	39.47%	92	60.53%	152
Total	157	52.33%	143	47.67%	300

$X^2 = 31.5$; P = 0.001

In study group, 52.3% had reactive NST and 47.6% had non – reactive NST. In patients with thick meconium stained liquor, 60.53% had non reactive NST.

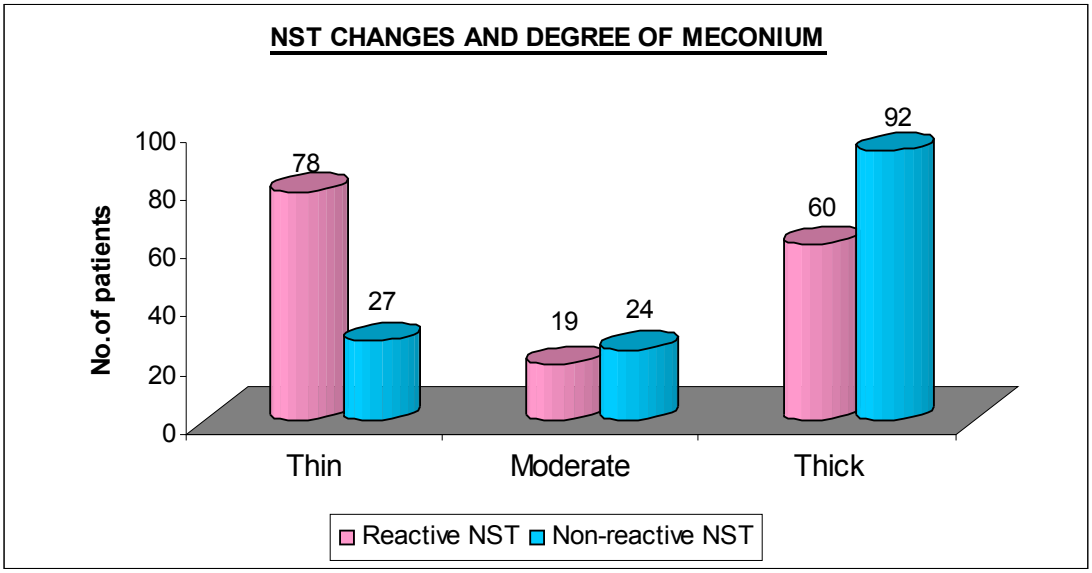

NST CHANGES AND DEGREE OF MECONIUM
No.of patients
100
80
60
40
20
0
78
27
19
24
60
92
Thin
Moderate
Thick
Reactive NST
Non-reactive NST

TABLE – 9

NST CHANGES IN NON – MECONIUM GROUP

NST	Number of patients	Percentage
Reactive NST	86	86%
Non reactive NST	14	14%

14% of the patients had non reactive NST.

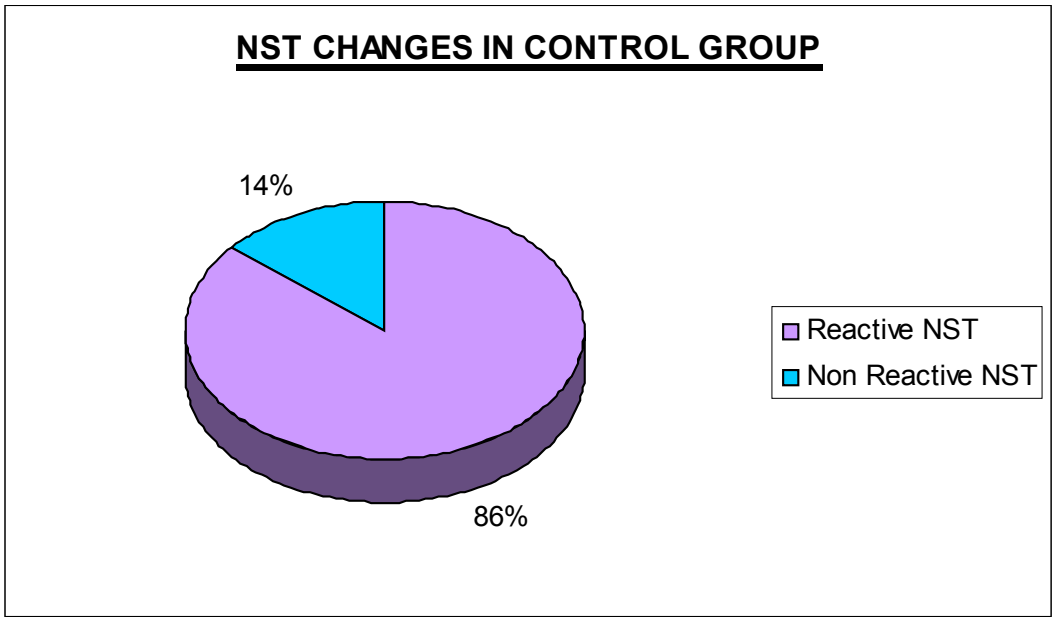

NST CHANGES IN CONTROL GROUP
14%
86%
Reactive NST
Non Reactive NST

TABLE – 10

MODE OF DELIVERY IN MECONIUM GROUP WITH RESPECT TO DIFFERENT DEGREES OF MECONIUM

Density of meconium	Labour Natural		LSCS		Forceps		VBAC		Total
	Patients	%	Patients	%	Patients	%	Patients	%	
Thin MSAF	65	61.9%	33	31.4%	7	6.6%	-	-	105
Moderate MSAF	13	30.2%	24	55.8%	5	11.6%	1	2.3%	43
Thick MSAF	44	28.9%	101	66.4%	3	1.9%	4	2.6%	152
Total	122	40.7%	158	52.7%	15	5%	5	1.7%	300

$X^2 = 42.3$; P = 0.001

In study group, 158 patients (52.7%) were delivered by LSCS, of which 101 patients had thick meconium stained liquor.

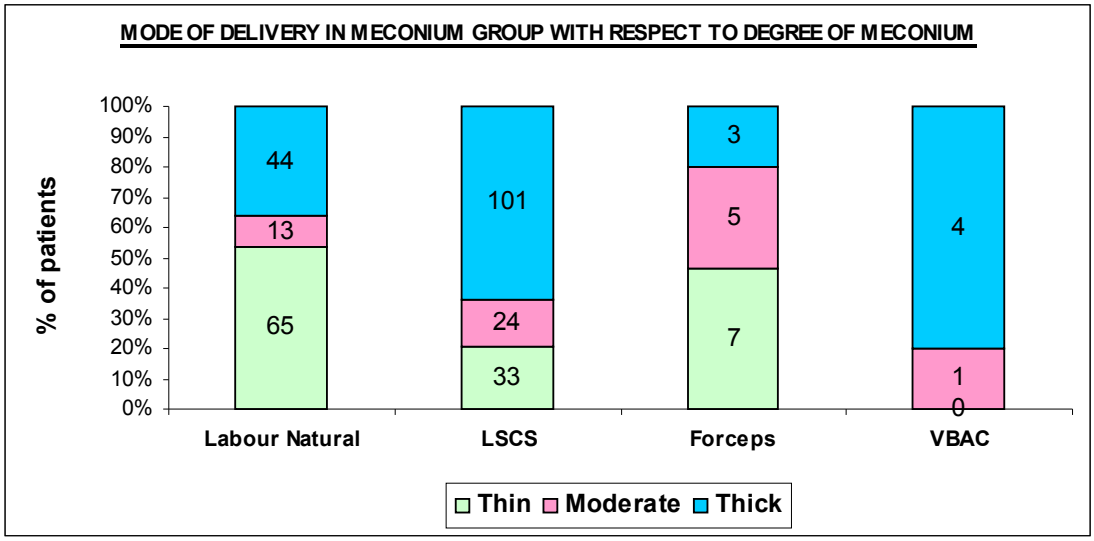

MODE OF DELIVERY IN MECONIUM GROUP WITH RESPECT TO DEGREE OF MECONIUM
% of patients
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
44
13
65
101
24
33
3
5
7
4
1
0
Labour Natural
LSCS
Forceps
VBAC
Thin
Moderate
Thick

TABLE - 11

MODE OF DELIVERY IN MECONIUM GROUP WITH RESPECT TO NST

Density of meconium	NST	Labour Natural		LSCS		Forceps		VBAC	
		Patients	%	Patients	%	Patients	%	Patients	
Thin MSAF	Reactive	52	66	24	30.8	2	2.6	-	
	Non reactive	13	48.2	9	33.3	5	18.5	-	
Moderate MSAF	Reactive	6	31.6	10	52.6	2	10.5	1	5
	Non reactive	7	29.2	14	58.3	3	12.5	-	
Thick MSAF	Reactive	15	25	43	71.6	-	-	2	3
	Non reactive	29	31.5	58	63	3	3.3	2	2
Total		122		158		15		5	

In thin meconium group, with reactive NST, 66% delivered vaginally. But in thick meconium group, irrespective of the NST, most of them were delivered by LSCS.

TABLE - 12

MODE OF DELIVERY IN NON – MECONIUM GROUP

Mode of delivery	**Number of patients**	**Percentage**
Labour natural	59	59%

LSCS	37	37%
Forceps	4	4%
VBAC	-	-

In the control group, majority 59% of the patients delivered by labour natural and 37% delivered by LSCS.

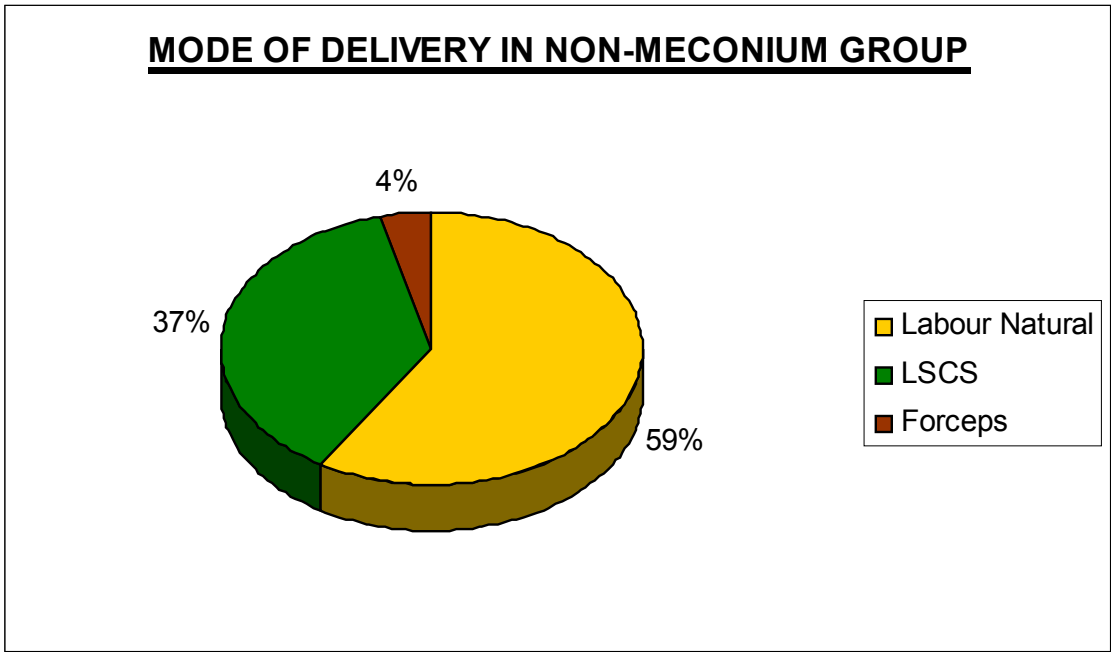

MODE OF DELIVERY IN NON-MECONIUM GROUP
4%
37%
59%
Labour Natural
LSCS
Forceps

TABLE - 13

MODE OF DELIVERY IN NON MECONIUM GROUP WITH RESPECT TO NST CHANGES

NST	Labour Natural		LSCS		Forceps		Total
	Patients	%	Patients	%	Patients	%	
Reactive	54	62.8%	28	32.6%	4	4.65%	86
Non Reactive	5	35.7%	9	64.3%	-	-	14
Total	59	59%	37	37%	4	4%	100

$X^2 = 5.4$; P = 0.07

Among 86 cases of reactive NST, 62.8% of cases delivered by labour natural. Among 14 cases of non reactive NST, 64.3% of cases delivered by LSCS.

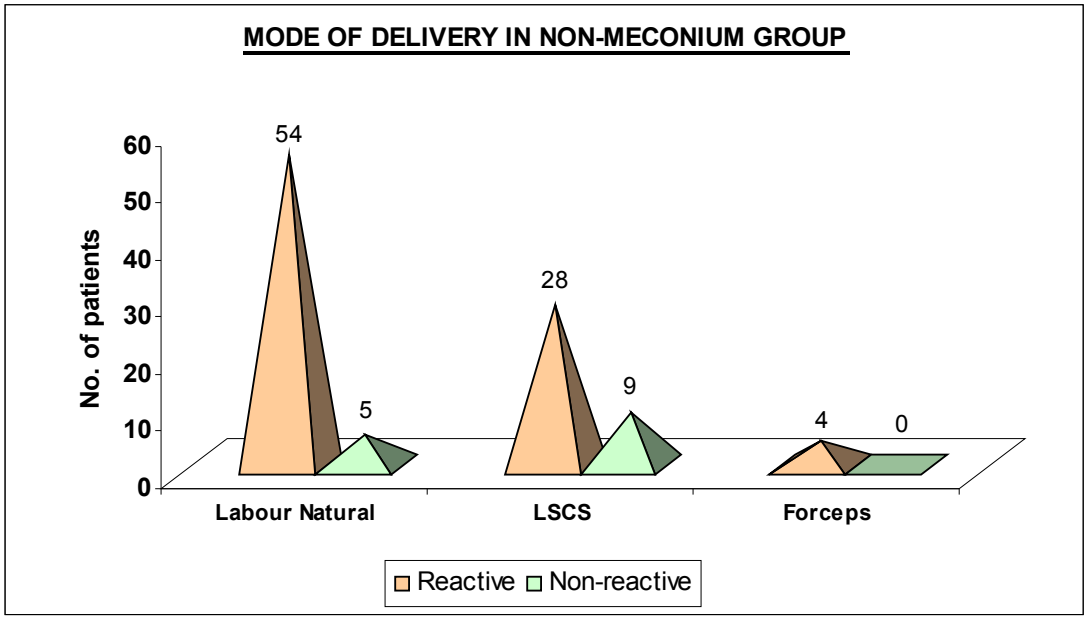

MODE OF DELIVERY IN NON-MECONIUM GROUP
No. of patients
60
50
40
30
20
10
0
54
5
28
9
4
0
Labour Natural
LSCS
Forceps
Reactive
Non-reactive

TABLE - 14

INDICATIONS FOR CESAREAN SECTION IN STUDY AND CONTROL GROUP

S.No.	Indications	Study Group	Control Group
1.	Fetal distress	111 (70.2%)	7 (18.9%)
2.	CPD	28	15
3.	CPD with fetal distress	5	-
4.	Persistent occipito posterior	1	2
5.	Failed induction	3	7
6.	Previous LSCS with PROM	6	1
7.	Other Causes	4	5
	Total No. of Patients	158	37

Among 158 patients, 31 patients had repeat LSCS in study group. In control group, 14 patients had repeat LSCS. The most common indication for LSCS in the study group is fetal distress, 70.2% vs 18.9% in the control group, Z = 33.4; P = 0.001; Odds Ratio = 10; 95% CI = 4 – 27.

TABLE - 15

APGAR SCORE AT 1 MIN IN MECONIUM GROUP WITH RESPECT TO DIFFERENT DEGREES OF MECONIUM

1 min Apgar	Thin MSAF		Moderate MSAF		Thick MSAF		Total
	Patients	%	Patients	%	Patients	%	
≥ 7/10	71	67.62	23	53.5	60	39.5	154 (51.3%)
5/10, 6/10	22	20.9	16	37.2	57	37.5	95 (31.7%)
≤ 4/10	12	11.4	4	9.3	35	23.0	51 (17%)
Total	105		43		152		300

$X^2 = 16.7$; P = 0.01

Using 1 min Apgar score, only 51.3% of babies had a good Apgar ≥ 7/10 at 1 min. In thick meconium, only 39.5% of babies had Apgar ≥ 7/10 at 1 min, and 23% of babies had a poor Apgar of ≤ 4/10 at 1 min.

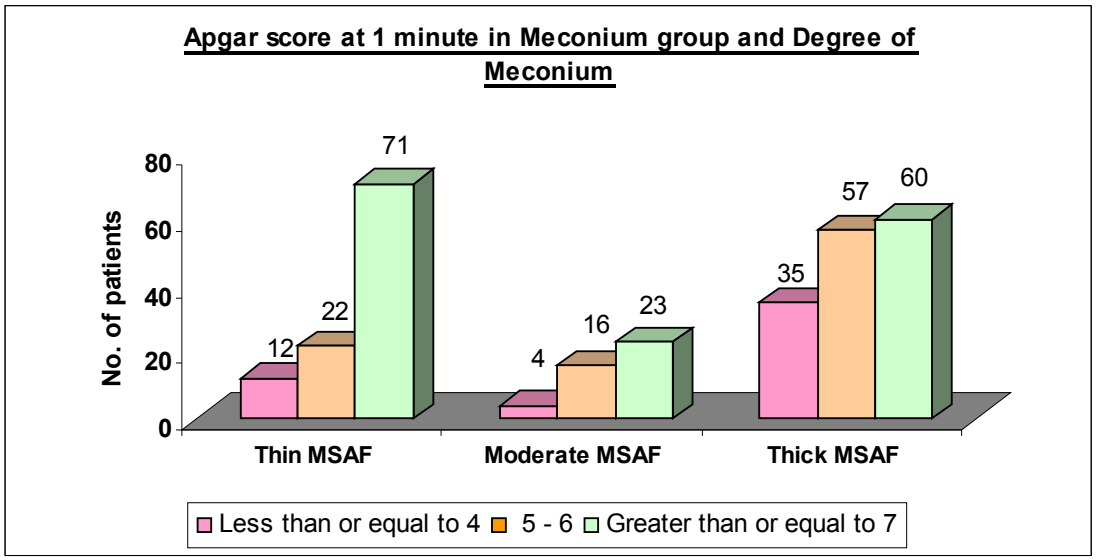

Apgar score at 1 minute in Meconium group and Degree of Meconium
No. of patients
80
60
40
20
0
12
22
71
4
16
23
35
57
60
Thin MSAF
Moderate MSAF
Thick MSAF
Less than or equal to 4
5 - 6
Greater than or equal to 7

TABLE - 16

APGAR SCORE AT 5 MINS IN MECONIUM GROUP WITH RESPECT TO DIFFERENT DEGREES OF MECONIUM

5 min Apgar	Thin MSAF		Moderate MSAF		Thick MSAF		Total
	Patients	%	Patients	%	Patients	%	
≥ 7/10	97	92.4	41	95.4	129	84.9	267 (89%)
5/10, 6/10	6	5.7	2	4.7	20	13.2	28 (9.3%)
≤ 4/10	2	1.9	-	-	3	1.9	5 (1.7%)
Total	105		43		152		300

$X^2 = 6.3$; P = 0.18

Using 5 min Apgar score, 89% of the babies had a good Apgar of ≥ 7/10 at 5 mins. Even in patients with thick meconium, 84.9% of babies had a good Apgar of ≥ 7/10 at 5 mins.

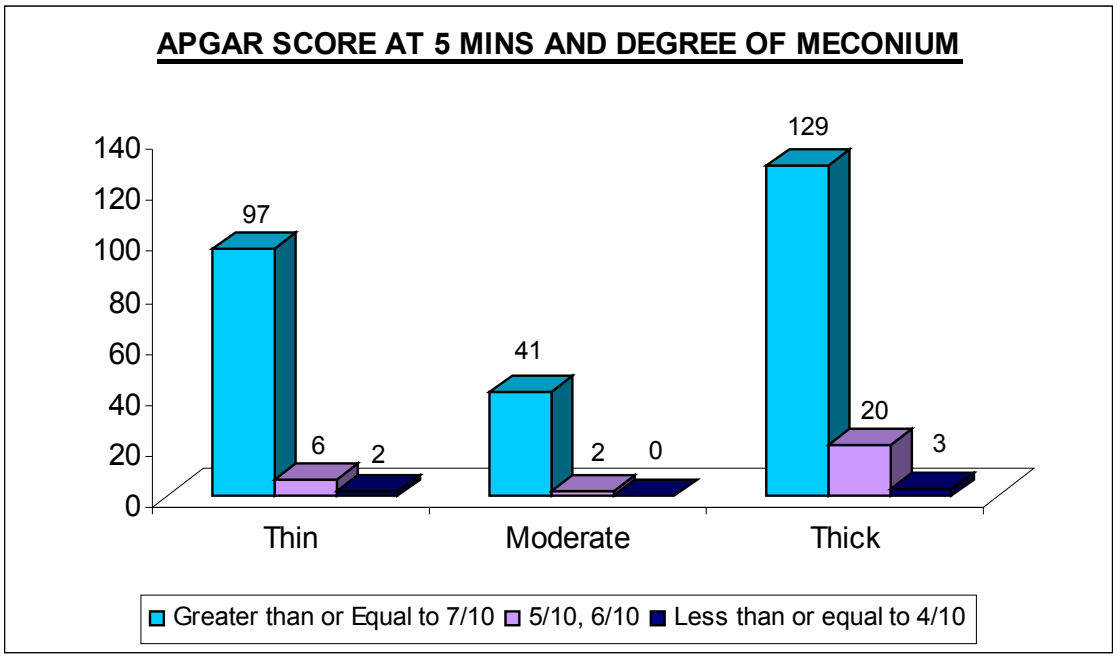


TABLE - 17

RELATIONSHIP BETWEEN DENSITY OF MECONIUM, NST AND APGAR SCORE

Density of meconium		≥ 7 /10		5/10, 6/10		≤ 4/10		Total	
		Patients	%	Patients	%	Patients	%		
Thin MSAF	Reactive	75	96.1%	2	2.6%	1	1.3%	78	X^2= 20.3 P= 0.001
	Non reactive	17	63%	7	26%	3	11%	27	
Moderate MSAF	Reactive	17	89.5%	2	10.5%	-	-	19	X^2= 0.04 P=0.84
	Non reactive	21	87.5%	3	12.5%	-	-	43	
Thick MSAF	Reactive	57	95%	3	5%	-	-	60	X^2= 29.2 P= 0.001
	Non reactive	50	54.3%	27	29.3%	15	16.3%	92	
Total		237	79%	44	14.6%	19	6.3%	300	

With reactive NST, Apgar score is good in all three groups. The incidence of low Apgar is higher in those with non – reactive NST. This is satistically significant in thin and thick meconium groups.

Table – 18

APGAR SCORE AT 5 MINS IN NON – MECONIUM GROUP WITH RESPECT OF NST

Apgar at 5 min	Reactive NST	Non reactive NST	Total
≥ 7 /10	86	13	99
5/10, 6/10	-	1	1
≤ 4/10	-	-	-
Total	86	14	100

All patients with reactive NST had a good Apgar of ≥ 7/10 at 5 mins.

TABLE - 19

APGAR SCORE AT 1 MIN IN NON – MECONIUM GROUP WITH RESPECT TO NST

Apgar at 1 min	Reactive NST	Non reactive NST	Total
≥ 7 /10	67	12	79
5/10, 6/10	19	2	21
≤ 4/10	-	-	-

About 79% of patients had a good Apgar of ≥ 7/10 at 1 min.

TABLE - 20

BIRTH WEIGHT OF BABIES IN MECONIUM GROUP

Birth weight	Thin MSAF	Moderate MSAF	Thick MSAF	Total
1.5 – 1.9 kg	1	-	8	9
2 – 2.4 kg	7	5	24	36
2.5 – 2.9 kg	36	17	47	100
3 – 3.4 kg	41	16	45	102

> 3.5 kg	20	5	28	53

$X^2 = 13.5$; P = 0.05

TABLE 21

BIRTH WEIGHT OF BABIES IN CONTROL GROUP

Birth weight	No. of Babies
< 2.5 kg	8
2.5 – 2.9 kg	37
3 - 3.5 kg	49
> 3.5 kg	6

Majority of the babies are in the birth weight range 2.5 to 3.5kg in both study and control group.

Many IUGR babies were noticed with, thick meconium stained liquor.

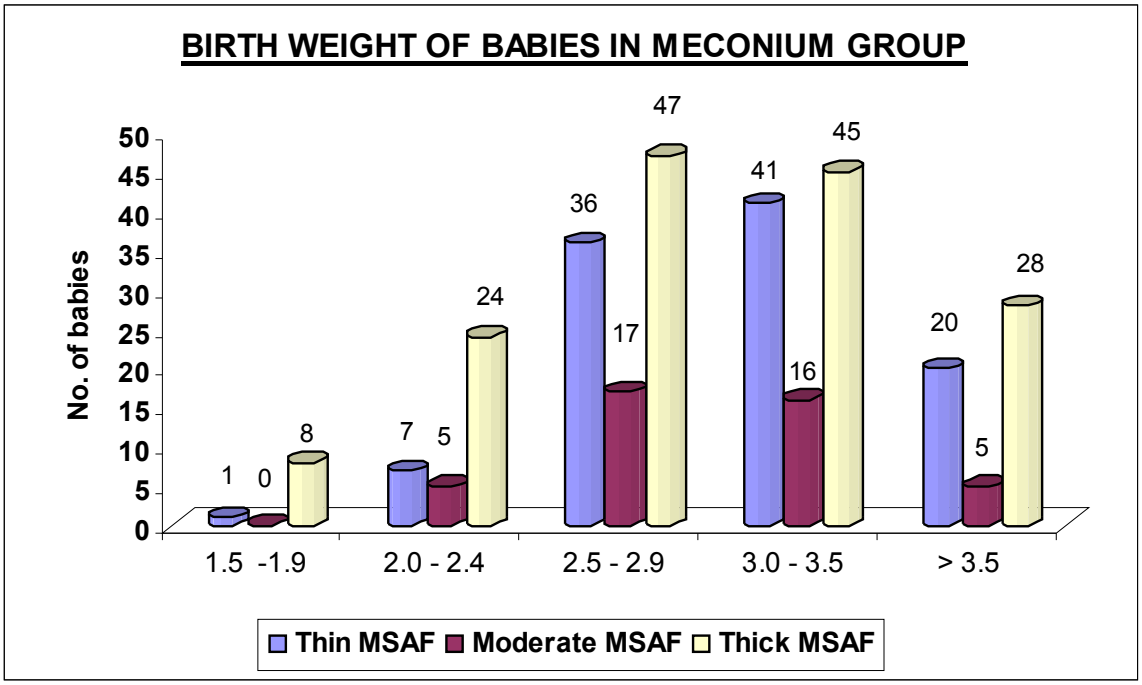

BIRTH WEIGHT OF BABIES IN MECONIUM GROUP
No. of babies
50
45
40
35
30
25
20
15
10
5
0
1
0
8
7
5
24
36
17
47
41
16
45
20
5
28
1.5 -1.9
2.0 - 2.4
2.5 - 2.9
3.0 - 3.5
> 3.5
Thin MSAF
Moderate MSAF
Thick MSAF

TABLE - 22

NICU ADMISSION IN MECONIUM GROUP WITH

RELATION TO NST

Density of meconium	Reactive NST		Non reactive NST		Total number of patients	
	Number of patients	%	Number of patients	%		
Thin MSAF	12	57.1%	10	13.5%	22	23.2
Moderate MSAF	2	9.5	8	10.8	10	10.5
Thick MSAF	7	33.3	56	75.7	63	66.3
Total	21	100	74	100	95	100

Total number of babies admitted = 95 (31.7%)

Among 157 reactive NST only 21 (13.4%) babies were admitted. Among 143 non reactive NST, 74 babies (51.8%) were admitted. Majority of the admission is constituted by babies from thick meconium group with non – reactive NST (60.9%).

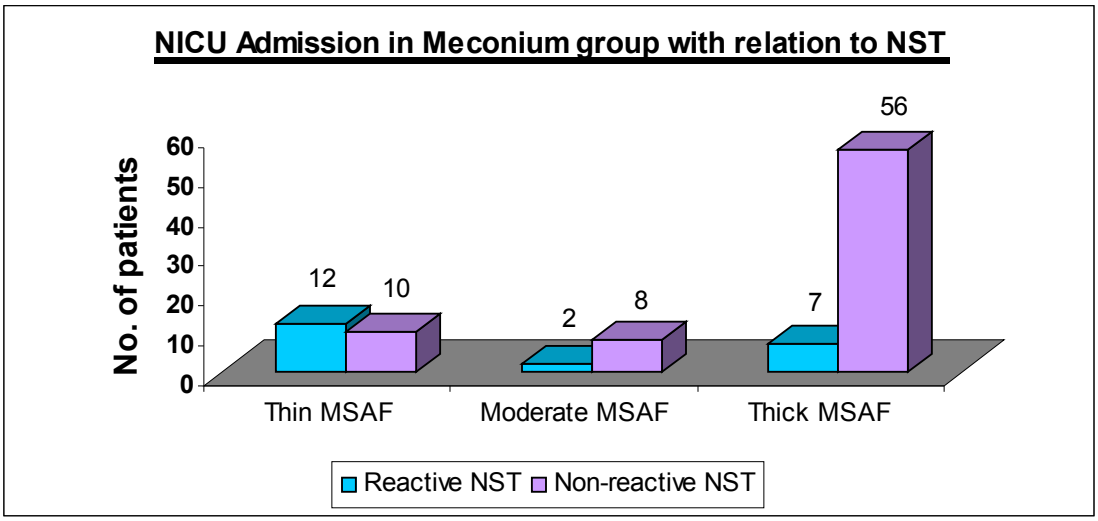

NICU Admission in Meconium group with relation to NST
No. of patients
60
50
40
30
20
10
0
12
10
2
8
7
56
Thin MSAF
Moderate MSAF
Thick MSAF
Reactive NST
Non-reactive NST

TABLE 23

REASON FOR NICU ADMISSION IN MECONIUM GROUP

S.No.	Reason for Admission	Number of babies admitted
1.	MAS	25
2.	IUGR with MAS	6
3.	LGA	5
4.	Respiratory distress	33
5.	IUGR	12
6.	HIE I and II	3
7.	Intraventricular hemorrhage	1
8.	Subgaleal bleed	1
9.	To R/O Sepsis	6
10.	IDM	3
Total		95

Percentage of MAS in this study = 10.3%

Among 6 babies which were admitted to TOR/O sepsis, sepsis was present in 3 babies.

TABLE 24

NICU ADMISSION IN CONTROL GROUP

NST	**Number of babies admitted**	**Percentage**
Reactive	4	4.7%
Non Reactive	1	7.1%
Total	5	5%

Among 86 reactive NST. Only 4.7% babies were admitted. Among 14 non reactive NST, 7.1% of babies were admitted.

TABLE 25

REASONS FOR ADMISSION IN CONTROL GROUP

S.No.	**Reason**	**Number of babies admitted**
1.	LGA	2
2.	IUGR	1
3.	IDM	1
4.	Perinatal hypoxia	1

	(NR NST)	

Perinatal mortality was nil with Control Group

TABLE 26

PERINATAL MORTALITY IN THE STUDY GROUP (MECONIUM)

Density of Meconium	**Reactive NST**	**Non reactive NST**	**Total**
Thin MSAF	-	3	3
Moderate MSAF	-	1	1
Thick MSAF	-	6	6
		Total	10 (3.3%)

Total number of neonatal deaths – 10 (3.3%). All neonatal deaths occurred in those patients with non – reactive NST. In patients with thin meconium stained liquor. 3 babies died – 2 babies died due to severe birth asphyxia and hypoxic ischemic encephalopathy stage II and 1 baby died due to meconium aspiration syndrome (IUGR). In patients with moderate meconium stained liquor, 1 baby died due to hypoxic ischemic encephalopathy II (IUGR). In patients with thick meconium stained liquor, 6 babies died. 4 babies died due to meconium aspiration syndrome, 1 baby died due to severe birth asphyxia and another one baby died due to bilateral pneumothorax, which developed as a sequelae

of MAS.

TABLE 27

COMPARISON OF PERINATAL MORTALITY IN STUDY AND CONTROL GROUPS

	Study Group	**Control Group**
Total number of patient	300	100
Neonatal deaths	10	0
Percentage	3.3%	0

Z = 1.36; P = 0.17

TABLE 28

COMPARISON OF PERINATAL MORTALITY WITH RELATION TO NST

	Study group		**Control group**	
	Reactive	**Non – Reactive**	**Reactive**	**Non – Reactive**
Total Number of patients	157	143	86	14
Neonatal	0	10	0	0

deaths				

Z = 3.02; P = 0.003 95% CI = 7% (2 – 12%)

The perinatal mortality becomes significant (P = 0.003) in meconium stained liquor, with non – reactive NST.

DISCUSSION

This prospective study consists of 300 patients with meconium stained amniotic fluid in labour as study group. The control group consists of 100 patients in labour with clear liquor. There were 30 cases of PIH, 7 cases of GDM and 112 cases of post dated pregnancy in study group. In control group there were 10 cases of PIH, 3 cases of GDM, and 35 cases of post dated pregnancy.

In the study group, 50.7% of patients had thick meconium stained liquor, 14.3% had moderate meconium and 35% of patients had thin meconium stained liquor.

In the study group, 47.6% of the patients had non reactive NST vs 14% in the control group, (P = 0.001). This shows that the incidence of non – reassuring fetal heart rate pattern was significantly higher in women with meconium stained amniotic fluid in labour (47.6% vs 14%).

Similar observation was made by **Starks et al., (1980).** They found significantly increased incidence of fetal heart rate abnormalities in the meconium group (32.7%) than control (6.1%).

NON REACTIVE NST IN STUDY AND CONTROL GROUP

	Study group	**Control group**
Present study	47.6%	14%
Starks et al (1980)	32.7%	6.1%

The reason for higher fetal heart rate abnormalities in the present study may be explained as follows : The criteria for FHR abnormalities in the present study are persistant tachycardia / bradycardia, absence of variability, repetitive late decelerations, whereas in Starks study, the criteria for FHR abnormalities was only late decelerations.

Similar observations were also made by ***Wong SF, Chow (2002).*** They remarked that the incidence of non – reassuring cardiotocography in women presenting with meconium stained amniotic fluid was significantly higher (9.8% vs 6.4%).

Depending upon the density of meconium, in patients with thick meconium stained liquor, majority (60.5%) had non – reactive NST and 39.5% had reactive NST. Similar observation was made by ***Halvax et al., (2002)*** that there is a significant linear association between the thickness of meconium and abnormal fetal heart rate pattern during labour.

In the study group, almost half of the patients (52.7%) were delivered by cesarean section as compared to 37% in the control group. (52.7% vs 37%). This correlate closely with the study conducted by ***Faridi, Aggarwal, Delhi (2004)*** where the incidence of Cesarean section in patients with meconium stained liquor is 48%.

CESAREAN SECTION INCIDENCE :

Faridi et al	48%
Present study	52%

Similarly, ***Ziadeh and Sunna (2000)*** also found that delivery by cesarean section increased by 7 -14% in patients with meconium stained liquor.

In a study conducted by ***Maymon, Chiam and Furman (1998)*** in low risk population, the incidence of cesarean section was 5.6% vs 2.3% ($P < 0.01$).

In this present study, 42% of the patients delivered vaginally, 52.7% of patients were delivered by cesarean section and 5% had instrumental delivery ($X^2 = 42.3$, $P = 0.001$).

The most common indication for cesarean section in the meconium group was fetal distress (70.2% vs 18.9% in control group); $P = 0.001$.

In study group, with reactive NST, 94.9% of babies had good mean Apgar score of ≥ 7/10. But with non reactive NST only 61.5% had a good mean Apgar score of ≥ 7/10. This shows that the

incidence of low Apgar score at 1 and 5 minutes in meconium stained amniotic fluid was significantly higher when associated with fetal heart rate abnormalities.

A number of investigators have associated low Apgar scores with meconium stained amniotic fluids ***(Starks, 1980; Cole et al, 1985; Ziadeh and Sunna, 2000).*** In these studies, the incidence of low 1 and 5 minute Apgar score was approximately two times greater when meconium was present.

Meis and associates (1978) found that heavy meconium increased the risk of low Apgar score, MAS and death compared with light meconium. But later on, ***Bochner and Co authors (1987)*** reported that even with thick meconium, perinatal morbidity was increased only when non reassuring FHR pattern were present.

In the study group, 31.7% of the babies were admitted for NICU care. In patients with reactive NST only 13.4% of the babies were admitted, but in those with non – reactive NST 51.8% of babies were admitted (P = .0001).

Similarly, many early reports have related meconium passage to increased risk of perinatal morbidity and mortality, especially when associated with abnormal fetal heart rate patterns. There were reports by ***Fenton*** and ***Steer, 1992; Hoble, 1971, Miller et al., 1975; Krebs et al, 1980; Nathan et al, 1994.***

Ash AK, Cambridge (2000) have suggested that meconium stained amniotic fluids might signify underlying acute or chronic fetal hypoxia with adverse perinatal outcome, especially when associated with cardiotocographic abnormalities.

The incidence of meconium aspiration syndrome in the study was 9.66%. This corresponds closely to the studies conducted by ***Davis et al., 1985; Falciglia 1988; Rossi et al., 1989;*** where they reported the percentage of MAS developing in infants delivered through meconium stained amniotic fluid is 8 – 10%.

In this present study, when MAS incidence is further classified depending upon the density of meconium, the MAS incidence in thick meconium stained liquor in 16.4%, in moderate meconium stained liquor 2.3% and in thin meconium stained liquor it is 3.8%. This correlates closely with the study done by ***Rossi and colleagues, 1989,*** where they reported MAS incidence of 19% in patients with thick meconium stained liquor, 5% in patients with moderate meconium stained liquor and 3% in patients with thin meconium stained liquor.

Incidence of MAS :

	Thin Meconium	**Moderate Meconium**	**Thick Meconium**
Rossi and Colleagues	3%	5%	19%
Present study	3.8%	2.3%	16.4%

The incidence of respiratory complications in our study is high among those group of patients with non reactive NST.

Fleischer and colleagues (1992) reported that the risk of neonatal respiratory complications in the presence of meconium was 2%, when FHR patterns were normal and 12% when they were abnormal.

Dellinger et al., (2000) retrospectively analysed intrapartum FHR patterns and reported that

outcomes such as cesarean section low Apgar and admission to the neonatal intensive care unit in patients with meconium stained liquor were significantly related to the FHR pattern.

The perinatal mortality in the study group was 3.3%. There was a total of 10 neonatal deaths. This corresponds closely to the study conducted by ***Zhao and Zhang, China (2000),*** where the perinatal mortality was 3.6% (n = 136, 5 neonatal deaths).

Perinatal mortality :

Zhao and Zhang, China	3.6%
Present study	3.3%

According to ***Ziadeh and Sunna (2000)*** Jordan University, the perinatal mortality increased from 2 per 1000 births with clear amniotic fluids to 10 per 1000 with meconium stained amniotic fluids P (< 0.001). According to Fraser*,* ***Hofmeyr and Alexander (2005)***, the perinatal mortality was 0.5%.

In this present study, with respect to NST, there was no neonatal deaths in those patients with reactive NST and there were 10 neonatal deaths in those with non – reactive NST. (7% vs 0) P = 0.003, 95% CI = 7% ranging from 2% to 12%. This again indicates that meconium stained amniotic fluid has a significant risk of causing perinatal mortality only when it is associated with FHR abnormalities.

Neonatal deaths in the present study :

Case No 28 :

25 years old, Selvi, $G_3P_2L_2$ with 40 weeks gestation (mild preeclampsia) was admitted in active labour. ARM was done. Had thick meconium stained liquor with non reactive NST. Amnioinfusion was given. Since estimated fetal weight was only 1.3kg and patient was in active labour, vaginal delivery was planned. Patient delivered in 40 minutes by Labour Natural. Baby weighing 1.5kg with Apgar 4/10, 7/10. Baby developed MAS and died on 2nd postnatal day.

Case No 30 :

28 years old, Ammu, $G_4P_3L_3$ with 40 weeks gestation was admitted in active labour. ARM was done. Had thick meconium stained liquor with non reactive NST. Amnioinfusion was given. Patient delivered in 25 minutes by labour natural. Admission – delivery interval was 40 mins. Baby weighing 3.2 kg with Apgar 1/10, 3/10, 4/10 – severe Birth Asphyxia. Baby was connected to ventilator and died 30 hours after birth.

Case No 40 :

21 years old, Revathy, a primigravida with 39 weeks gestation was admitted in active labour. ARM was done. Had thin meconium stained liquor with non reactive NST. Since she was in active labour with an estimated fetal weight of 1.8kg, vaginal delivery was planned. Delivered by LMC forceps in 25 minutes. Baby weighing 2.2 kg with Apgar 3/10, 5/10 – severe birth asphyxia. Baby was connected to ventilator and died 48 hours after birth.

Case No 47 :

26 years old, Dhanalakshmi, G_2A_1 with 38 weeks gestation was admitted in active labour with thin meconium stained liquor draining PV. NST was non reactive. Patient was delivered by LMC

forceps in 45 minutes. Baby weighing 2.7 kg with Apgar 3/10, 4/10, 5/10- Perinatal hypoxia. Baby developed hypoxic ischemic encephalopathy stage II and died 60 hours after birth.

Case No 59 :

23 years old, Subashini, a primigravida with 40 weeks gestation was admitted in active labour. ARM was done. Had moderate meconium stained liquor with non reactive NST. Since the estimated fetal weight was 1.6kg, vaginal delivery was planned. She delivered by labour natural in 30 minutes. Baby weighing 2 kg with Apgar 3/10, 6/10. Baby developed hypoxic ischemic encephalopathy stage II and died 36 hours after birth.

Case No 112 :

Komala, 25 years old, G2A1 with 40 weeks 5D gestation was admitted in active labour. ARM was done. Had clear liquor with non – reactive NST. She delivered by labour natural within 30 minutes of detecting non reactive NST. Hindwaters was thin meconium stained. Baby weighing 3.1 kg with Apgar 1/10, 3/10, 5/10. Baby developed hypoxic ischemic encephalopathy stage II and died 32 hours after birth.

Case No 136 :

Nirmala, 25 years old primigravida with 39W5D gestation had high fever on admission (Temp – 101^{o}F). Patient was in active labour. ARM was done. Had thick meconium stained liquor with non reactive NST. Amnioinfusion was given. She delivered in 20 minutes by labour natural. Baby weighing 3 kg with Apgar 3/10, 5/10, 6/10. Baby developed MAS and died 60 hours after birth.

Case No 152 :

23 years old, Sreedevi, $G_2P_1L_0$ with 38 weeks gestation was admitted with Jaundice complicating pregnancy and she was in active labour with thick meconium stained liquor draining PV. NST was non reactive. Amnioinfusion given. She delivered by labour natural in 40 minutes. Baby weighing 2.8 kg, with Apgar 3/10, 5/10. Baby developed MAS and died on 3rd postnatal day.

Case No 173 :

23 years old, Jayalaxmi, G_4A_3 with 39 weeks 3D gestation was admitted in early labour with draining PV. Had thick meconium stained liquor and non reactive NST. Amnioinfusion was given. Emergency LSCS was done in 30 minutes. Baby weighing 2 kg with Apgar 2/10, 4/10, 5/10. Baby developed MAS and died 48 hours after birth.

Case No 239 :

24 years old, sangeetha, G_2 A_1 with 39 weeks gestation was admitted in early labour with thick meconium stained liquor draining PV. NST was non reactive. Amnioinfusion given. Emergency LSCS was done within 30 minutes. Baby weighing 2.8 kg with Apgar 4/10, 6/10, 6/10 - severe perinatal hypoxia. Baby developed MAS and Bilateral pneumothorax and died on 4th postnatal day.

SUMMARY

- This prospective study was conducted to evaluate the perinatal outcome in meconium stained amniotic fluid. The study group consists of 300 patients in labour with meconium stained amniotic fluid. Among these patients, 50.7% had thick meconium stained liquor, 35% of the patients had thin meconium stained liquor and 14.3% of the patients had moderate meconium stained liquor. The control group consists of 100 patients in labour with clear liquor.

- Meconium stained liquor was detected in the latent phase in 59% of the patients. Most of the patients (64.3%) in the study group were delivered within 1 hour of detection of meconium.

- In the study group, 52.3% of patients had reactive NST and 47.6% had non reactive NST. Whereas in the control group, 86% of patients had reactive NST and only 14% had non – reactive NST. This shows that the incidence of non reassuring cardiotocography is significantly higher in women with meconium stained liquor in labour (47.6% vs 14%) P = 0.001.

- In thin meconium group of patients, majority (74.2%) of the patients had reactive NST. Whereas in patients with thick meconium stained liquor, majority (60.5%) had non – reactive NST. This indicates a significant linear association between the thickness of meconium and abnormal fetal heart rate pattern during labour.

- In the study group, almost half of the patients were delivered by cesarean section (52.7% vs 37% in the control group) P = 0.001. This higher rate of cesarean section in the meconium group is mainly contributed by patients in thick meconium group with non – reactive NST (36.7%). Fetal distress is the commonest indication for cesarean section in the study group (70.2% vs 18.9% in control

group) p = 0.001. There was 31 cases of repeat LSCS in the study group and 14 cases of repeat LSCS in the control group.

- In the meconium group with reactive NST 94.9% of babies had good mean Apgar score of ≥ 7/10 at 1 and 5mins. With non – reactive NST, only 61.5% of babies had good mean Apgar score of ≥ 7/10. In other words, 39.5% of babies had meand Apgar score of < 7/10. This shows that the incidence of low Apgar score at 1 and 5 minutes in meconium stained amniotic fluid was significantly higher when associated with fetal heart rate abnormalities. 39.5% vs 4.1%.

- In the study group, 31.7% of the babies were admitted for NICU care. In patients with reactive NST only 13.4% of the babies were admitted, but in those patients with non – reactive NST 51.8% of the babies were admitted to NICU. (51.8% vs 13.4%); P = 0.001. Majority of the NICU admission is constituted by babies from thick meconium group with non – reactive NST (58.94%).

- The commonest reason for NICU admission was mild respiratory distress (34.7%). The second most common reason was meconium aspiration syndrome (32.6%). The incidence of meconium aspiration syndrome in the study group was 10.3%. In the control group, only 5% of the babies were admitted.

- The perinatal mortality in the study group was 3.3% as compared to no neonatal death in the control group. (3.3% Vs 0); P = 0.17. There were total of 10 neonatal deaths. 3 babies died in the thin meconium group, 1 baby died in moderate meconium group and 6 babies died in the thick meconium group. All neonatal deaths has occurred in those patients with non reactive NST (7% Vs0) P = 0.003, 95% CI = 7%, (2 – 12%).

CONCLUSION

- The incidence of non reassuring fetal heart rate pattern is significantly higher in women with meconium stained amniotic fluid in labour. There is a significant linear association between the thickness of meconium and abnormal fetal heart rate pattern during labour.

- The perinatal outcome is good in patients with meconium stained amniotic fluid and reactive NST.

- The cesarean section rate, low Apgar Score, neonatal admissions and perinatal mortality were significantly higher with meconium stained amniotic fluid and non reactive NST.

So, meconium in the amniotic fluid is associated with obstetric hazard and significantly increased risk of adverse neonatal outcomes, only when it is associated with fetal heart rate abnormalities. The main clinical value of meconium stained amniotic fluid is to alert the obstetrician to look for further signs of fetal compromise.

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PROFORMA

Name : Age: IP No.: Unit:

Address:

Socio economic status: Booked/Unbooked:

Date and Time of Admission:

Initial Admission: ANWard/Labour ward/LW Annex:

Gravida Para Live Abortion LMP

Gestational age: EDD:

Obstetric history: M/H :

History suggestive of PIH/anaemia/heart disease/GDM/Post dated/APH:

Time of onset of labour:

Referred as Meconium stained liquour: Yes/No

General Examination:

Nutrition: Height:

Anemia: Weight:

Pedal edema :

Vital Signs: BP:

PR:

Temp:

CVS:

RS:

Obstetric examination:

P/A: P/V:

Any clinical evidence of postdatism/IUGR/Oligohydramnios:

Blood group:

Sonographic findings:

Labour onset: Spontaneous/Induced (cerviprime/synto)

Syntocinon for acceleration :Given / not given

Any other medicine used:

Duration of labour: Stage I II

Rupture of membranes: Spontaneous/ARM

If ARM, Date and time of ARM:

Colour of liqour:

Quantity of liquor:

Any FHR variation seen, NST:

Amnioinfusion: Given/not given

If meconium stained liqour

Time of detection of meconium, which stage of labour:

Induction - delivery interval:

Time interval between detection of meconium and delivery:

Mode of delivery : ☐ Labour Natural

☐ Instrumental

☐ Cesarean

If instrumental, indication:

If cesarean, indication:

Baby: Alive/Dead born

Sex: Male/female

Birth wt:

Apgar : 1 min 5 mins

Placenta at birth : Normal/calcification/ meconium stained/infarcts

Any evidence of IUGR/Postmaturity in the baby :

Other details:

Resuscitation methods:

Meconium at the level of vocal cords: Yes/No

NICU Admission : Yes/No

Reason for Admission: Observation/MAS/Birth asphyxia/
HIE/Respiratory distress/IUGR

Duration of NICU stay:

Treatment given:

Condition on discharge from PTU:

If died, cause and time of death:

ABREVATIONS

W	-	Weeks
D	-	Days
L	-	Latent Phase
A	-	Active Phase
R	-	Reactive Non – Stress Test
NR	-	Non – Reactive Non Stress Test
FHR	-	Fetal Heart Rate
LN	-	Labour Natural
LMC	-	Low Midcavity forceps
LSCS	-	Lower Segment Cesarean Section
FD	-	Fetal Distress
FI	-	Failed Induction
PROM	-	Premature rupture of membranes
CPD	-	Cephalo Pelvic Disproportion.
POP	-	Persistent Occipito Posterior
Pl. Previa	-	Placenta Previa
BOH	-	Bad Obstetric History
VBAC	-	Vaginal Birth After Cesarean Section
NICU	-	Neonatal Intensive Care Unit
LGA	-	Large for Gestational Age
IUGR	-	Intra Uterine growth Retardation
IVH	-	Intraventicular Haemorrhage
PIH	-	Pregnancy Induced Hypertension
GDM	-	Gestational Diabetes Mellitus

HIE	-	Hypoxic Ischemic Encephalopathy
R.D.	-	Respiratory Distress
IDM	-	Infant of Diabetic Mother
ROP	-	Right Occipito Posterior
Oligo	-	Oligohydramnios
B	-	Booked
UB	-	Unbooked